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Transient Ischemia as a Possible Etiology for Ventricular Dysfunction

In the interesting study presented by Tsuchihashi et al. (1), we believe a few points deserve further discussion. Myocardial damage as evidenced by the finding of a positive creatinine kinase in 56% and the more sensitive troponin T in 72% of patients supports a possible role for myocardial ischemia, even if transient, as an important contributor in the pathogenesis of the ventricular dysfunction observed. Although coronary vasospasm was considered, only 55% of the patients were tested for it. Because of glagovian remodeling, coronary arteries may harbor significant amounts of arteriosclerotic plaque without obvious stenosis in angiography. These diseased but near normal looking coronary segments may develop marked spasm upon stimulation (2). Additionally, multiple drugs may induce vasospasm in normal coronary arteries. Clasically, cocaine use might trigger intense vasospasm (3) besides other adverse cardiac effects like acceleration of atherosclerosis or direct cardiotoxicity. Various commonly used drugs including antimigraine medications such as ergotamine or sumatriptan may also cause coronary vasospasm (4,5). Therefore, drug and toxicology testing during the acute episode to exclude exposure to these agents might have been rewarding. With the growing worldwide epidemic of substance abuse and self-medication, physicians will more frequently encounter adverse cardiovascular events in "low risk" populations. If not correctly diagnosed, unrecognized and/or recurrent use may lead to life-threatening complications.

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REPLY

We appreciate the interest of Castro et al. in our recent clinical study on a heart syndrome with transient left ventricular (LV) apical ballooning without coronary artery stenosis mimicking acute myocardial infarction (AMI) (1). As first reported by Satoh et al. (2) and Dote et al. (3) and as pointed out by Castro et al., coronary vasospasm under various underlying disorders, including administration of adrenergic drugs, might be considered as an initial etiological basis of this novel syndrome. However, we defined this syndrome as: 1) suspected AMI based on persistent chest symptoms or electrocardiogram (ECG) changes (ST-T changes, abnormal Q-wave formation); 2) transient LV ballooning confirmed by left ventriculography (LVG) and/or echocardiography (which is generally mismatched with the magnitude of creatine kinase release and with the area perfused by a single coronary artery); and 3) confirmed normal epicardial artery (luminal narrowing of $<50\%$ in all three coronary arteries) within 48 h of onset. Actually, no case exhibited vasospasm during manifestation symptoms or ECG changes. Also, autopsy findings in some cases were different from those of myocardial ischemia (4). Therefore, the possibility of transient ischemia including vasospasm as a initiating factor of this syndrome could not be ruled out; however, we speculate that vasospasm is not a main cause. Important etiologic bases suspected from our study will be vigorous stress (cathecholamine exposure) (5,6), dynamic midventricular obstruction due to basal hypercontraction (7), and/or secondary myocardial ischemia by apical ballooning (increased wall tension). However, as already mentioned in the discussion (1), our study was a retrospective investigation, and there are several limitations. Further cases therefore should be investigated to determine regional and racial differences.

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